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Evaluation of P wave axis in distinguishing anatomical site of atrial septal defect

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It has been previously proposed that a method of distinguishing sinus venosus atrial septal defects from ostium secundum atrial septal defects is a leftward axis deviation (less than +30°) of the P waves on the electrocardiograms of patients with sinus venosus defects. The electrocardiograms of 150 patients with surgically corrected atrial septal defects were reviewed to test this proposition. The preoperative P wave axis was not useful in predicting the anatomical location of atrial septal defects.

Anatomically, atrial septal defects may be classified into three types. These are: (1) ostium secundum defects in which the defect is in the region of the fossa ovalis but may extend downward to lie astride the inferior vena cava; (2) sinus venosus defects in which the defect underlies the superior caval orifice, there is no margin of atrial septum superior to the defect, and there is often (if not invariably) partial anomalous venous connexion of right pulmonary veins to the superior vena cava and right atrium; and (3) ostium primum defects in which the defect overlies the ventricular septum and its attached, often malformed, atrioventricular valves.

In the early stages of intracardiac surgery, it was important to distinguish between the varieties of atrial septal defect (Lewis and Taufic, 1953; Lewis *et al.*, 1955; Bedford, 1960). Study of patients clinically, haemodynamically, and electrocardiographically provided the means to separate ostium primum from ostium secundum atrial septal defects. One of the most useful signs was left axis deviation of the QRS on the electrocardiogram of patients with primum defects (Braudo *et al.*, 1954; Walker *et al.*, 1956; Witham and Ellison, 1957; de Oliveira and Zimmerman, 1958; Barboza, Brandenburg, and Swan, 1958; Pryor, Woodwark, and Blount, 1959; Liebman and Nadas, 1960; Abramson and Burton, 1962) – a finding only rarely encountered in secundum defects (Harrison and Morrow, 1963).

In 1868 Wagstaffee (quoted by Hudson, 1955) described two cases of free communication between the atria due to a deficiency of the upper part of the atrial septum. Hudson in 1955 referred to this variety of atrial septal defect as a persistence of the vestibule of the sinus venosus, and the term 'sinus venosus defect' is still applied. Each large surgical series contains many cases with such defects, and the surgical techniques for their closure have been well described (Swan *et al.*, 1957; Lewis, 1958; Cooley, Ellis, and Bellizzi, 1961; Mustard *et al.*, 1961; Friedli *et al.*, 1972). Radiographically and at cardiac catheterization it is often possible to demonstrate or at least suspect these defects by: (a) localized distension of the superior vena cava above the right atrium; (b) angiocardiograms showing partial anomalous pulmonary venous connexion to the superior vena cava; (c) an increase in oxygen saturation in the low superior vena cava; and (d) entry of the catheter into a right pulmonary vein from the superior vena cava (though a secundum defect may have partial anomalous venous connexion to the superior vena cava). Clinically the sinus venosus defect is indistinguishable from a secundum defect.

Hancock (1964) noted that there was a high frequency of left axis deviation of the P waves (a P axis of less than +30° using the standard triaxial reference figure) on the electrocardiogram of patients with sinus venosus defect and persistent left superior vena cava. He proposed that this finding should suggest a sinus venosus defect if there were other clinical features of an atrial septal defect. This suggestion is cited in one of the recent texts of congenital heart disease (Perloff, 1970). Our experience has caused us to doubt this observation of Hancock and prompted this study.

Subjects and methods

Preoperative electrocardiograms of 150 patients who had undergone surgical closure of atrial septal defect were

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TABLE 1 *Age and sex distribution of the patients studied*

Type of defect	*Male*	*Female*	*< 10 years*	*> 10 years*	*Total*
Secundum atrial septal defect	37	82	58	61	119
Sinus venosus defect	9	10	4	15	19
Primum atrial septal defect	6	6	9	3	12
	52	98	71	79	150

TABLE 2 *Associated cardiac defects*

Type of Defect	*Persistent ductus arteriosus*	*Pulmonary stenosis*	*Mitral stenosis*	*Mitral insufficiency*	*Ventricular septal defect*
Primum atrial septal defect	0	1	0	10	0
Secundum atrial septal defect	4	7	1	4	1
Sinus venosus defect	0	1	0	0	0

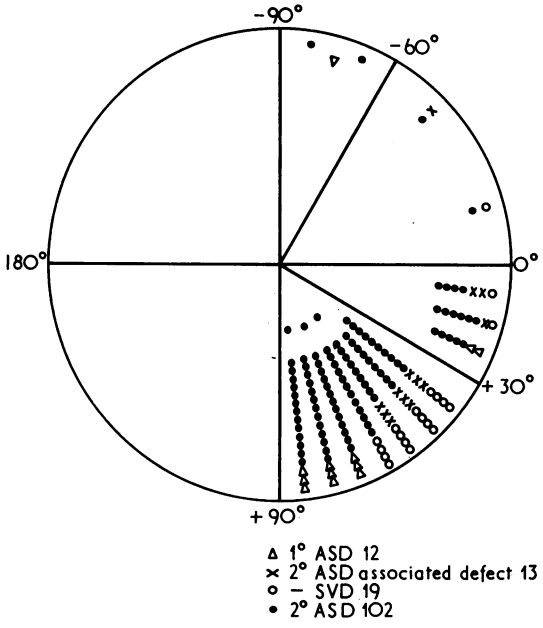

FIG. *Preoperative P wave axis of the patients with atrial septal defect in degrees as plotted on the standard hexaxial reference figure. 1° ASD = ostium primum septal defect. 2° ASD isolated = secundum atrial defect without associated anomaly. 2° ASD with associated defect = those secundum defects with partial anomalous pulmonary venous connexion or persistent left superior vena cava. SVD = sinus venosus defects. Three patients with secundum second-degree atrial septal defect and one patient with secundum second-degree atrial septal defect and associated defect had a shifting atrial pacemaker and are not shown on the above figure.*

examined. Anatomical location of the atrial defect in each case was determined from the operative note. Defects described as 'high atrial septal defects' were included in the secundum group unless there was associated partial anomalous venous connexion to the superior vena cava. The series contained 119 secundum defects, 19 sinus venosus defects, and 12 primum defects. More complex endocardial cushion defects (except when mitral insufficiency was the sole accompaniment of a primum defect) were excluded. The age and sex distribution of the patients is listed in Table 1. Other associated congenital heart defects are listed in Table 2.

All available electrocardiograms for each patient were examined and the preoperative P wave axis determined. The P axis was plotted on the standard hexaxial reference figure.

Findings

The preoperative P axis data are shown in Fig. 1. Patients with left axis deviation of the P waves on a given electrocardiogram were often noted to have other tracings with a normal P axis. In such cases the most leftward P axis was used. The observation that P axis can vary between tracings agrees with that of Hancock (1964).

The PR interval was normal or prolonged in all tracings used for tabulation of data, and in no case did the P wave morphology suggest left atrial hypertrophy. Three patients with primum defects and one patient with a secundum defect with left axis deviation of the P waves had an associated mitral valvular lesion.

Of the 119 patients with secundum defects, 23 had a P axis of less than +30° (22%). Of this group with left axis deviation of the P waves, 1 had partial anomalous venous connexion and 3 had persistent left superior vena cava. Within the total of 119 secundum defects, 10 patients had what was de-

scribed as a 'high atrial septal defect' (without associated defects). Three of these patients had left axis deviation of the P waves and 1 had a shifting atrial pacemaker. A shifting atrial pacemaker was present in a total of 4 patients with secundum defects. One of these patients had persistent left superior vena cava.

Nineteen patients had a sinus venosus defect. Three (16%) of these had left axis deviation of the P waves (of which 1 also had persistent left superior vena cava). There were no patients with sinus venosus defect who had a shifting atrial pacemaker.

Twelve patients had ostium primum defects. Three of these patients had left axis deviation of the P waves (25%).

Postoperatively the P axis would frequently shift to the right or left. This phenomenon has been noted by others (Mustard *et al.*, 1961; Hancock, 1964; Sealy *et al.*, 1969) and attributed to interruption of the atrial, internodal pathways of conduction (Sealy *et al.*, 1969).

Discussion

The incidence of inverted P waves in lead III has been reported as 5 per cent in normal adults and 10 per cent in normal children (Shipley and Hallaran, 1936; Burnett and Taylor, 1936). Most of the early papers discussing the electrocardiographic features of atrial septal defects did not comment on P axis (Braudo *et al.*, 1954; Witham and Ellison, 1957; Pryor *et al.*, 1959; Liebman and Nadas, 1960; Abramson and Burton, 1962; Harrison and Morrow, 1963; Sánchez Cascos and Deuchar, 1963). Barboza *et al.* (1958) found 10 (11%) of 90 patients with proven atrial defects had left axis deviation of the P waves. de Oliveira and Zimmerman (1958) noted that 7 (18%) of their 44 patients with atrial defects had left axis deviation of the P waves and attributed this leftward shift of P axis to 'left atrial overload' (mitral valve disease). Hancock (1964) was the first to attach significance to a left axis deviation of P waves in patients with atrial defects. In his series, 8 of 10 patients with either sinus venosus defect or high atrial defect associated with persistent left superior vena cava, and 14 of 20 patients with persistent left superior vena cava without atrial defect had a P axis of less than $+30°$; whereas, only 3 of 62 patients with secundum defect had a similar P axis. He felt that this shift of P axis was due to an ectopic pacemaker in the region of the coronary sinus and that the embryology of sinus venosus defect and persistent left superior vena cava predisposed these lesions to such a shift. He felt that a P axis of less than $+30°$ represented coronary sinus rhythm. He based his conclusions on the following observations: (a) one of his patients with sinus venosus defect had a shift of atrial pacemaker during a single tracing, suggesting that two separate atrial pacemakers were present; (b) patients with sinus venosus defects would often develop left axis deviation of their P waves postoperatively; and (c) the frequent occurrence of left axis deviation of P waves in patients with persistent left superior vena cava (which drains into the coronary sinus).

Patten noted the presence in embryonic hearts of bilaterally symmetrical pacemakers at the junction of the common cardinal veins with the right and left horns of the sinus venosus. He suggested that the adult SA node developed from the right cardinal vein pacemaker, and the AV node developed from the left cardinal vein pacemaker (Patten, 1956). Since persistent left superior vena cava represents persistence of the embryonic left cardinal vein, it may be that the left pacemaker may also persist in abnormally active form and function as a pacemaker much like the SA node.

Numerous theories have been proposed for the embryology of the sinus venosus defect and they have been reviewed by Harley (1958). In essence, the septum secundum is defective superiorly, and this explains the presence of the high atrial defect. The means by which the pulmonary veins attach to the atriocaval junction are not clear. The high atrial defect is at the site normally occupied by the SA node and is one of the reasons why Hancock presumed an ectopic pacemaker was often present.

Some authors doubt that the area of the coronary sinus contains tissue capable of spontaneous pacemaker activity (James, 1970). Classically, coronary sinus rhythm is recognized by inverted P waves in leads II, III, and aVF (P axis of less than $-30°$) associated with a normal or prolonged PR interval. Such a rhythm can be obtained by artificial pacing at the orifice of the coronary sinus (Leon *et al.*, 1970; Lancaster *et al.*, 1965), but it can also be obtained by pacing of the posterior left atrium (Harris *et al.*, 1968; Waldo *et al.*, 1970). The term coronary sinus rhythm, as described, is not specific and probably should be replaced by the term inferior atrial rhythm or ectopic atrial pacemaker (Piccolo, Nava, and Volta, 1971).

The data presented here do not agree with those of Hancock nor are his observations substantiated. Our incidence of left axis deviation of the P waves with sinus venosus defect and/or persistent left superior vena cava is less than in secundum defect (with or without persistent left superior vena cava) and is not much greater than the incidence of such a phenomenon in published series of atrial septal

defect (de Oliveira and Zimmerman, 1958; Barboza *et al.*, 1958). Furthermore, the incidence of shifting atrial pacemaker was far greater in the patients with secundum defect in our series than in those patients with sinus venosus defects. The embryological theory explaining the existence of an ectopic pacemaker in the region of the coronary sinus producing left axis deviation of P waves is attractive, but a similar P wave may be generated by pacemakers located in a variety of sites throughout the atria and is not specific for origin from the coronary sinus. Hancock's observation that the ectopic atrial pacemaker tends to appear and disappear is supported by this work but also has been noted to occur in patients without any form of heart disease (Lamb, 1965). Contrary to the work of Hancock, it is our observation that the tendency of P axis to shift after operation is not restricted to sinus venosus defect (though a leftward shift was more common after operation for sinus venosus defect than for secundum defect). This also has been the observation of others (Mustard *et al.*, 1961; Hancock, 1964; Sealy *et al.*, 1969). The suggestion of the absence of a normal SA node and the necessity for an ectopic atrial pacemaker is supported by the high incidence (75%) of extreme left axis deviation of P waves (less than $-45°$) in patients with a single atrium (Muñoz-Armas *et al.*, 1968), but such extreme left axis deviation of P waves was not common in this series or that of Hancock.

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